


Sodium restriction improves nocturia in patients at a cardiology clinic

Thomas F. Monaghan BS¹  | Kyle P. Michelson BA¹ | Zhan D. Wu BS¹ | Fred Gong BA¹ | Christina W. Agudelo MA¹ | Christopher D. George BA¹ | Upeksha S. Alwis MS² | Matthew R. Epstein MD¹ | Pakinam Mekki BS¹ | Viktor X. Flores MD¹ | Donald L. Bliwise PhD³ | Karel Everaert MD, PhD² | Johan Vande Walle MD, PhD⁴ | Jeffrey P. Weiss MD¹ | Jason M. Lazar MD, MPH⁵

¹Department of Urology, State University of New York Downstate Health Sciences University, Brooklyn, NY, USA

²Department of Urology, Ghent University Hospital, Ghent, Belgium

³Department of Neurology, Emory University School of Medicine, Atlanta, GA, USA

⁴Department of Pediatric Nephrology, Ghent University Hospital, Ghent, Belgium

⁵Division of Cardiovascular Medicine, Department of Medicine, SUNY Downstate Health Sciences University, Brooklyn, NY, USA

Correspondence

Thomas F. Monaghan, BS, Department of Urology, SUNY Downstate Health Sciences University, 450 Clarkson Avenue, Box 79, Brooklyn, NY 11203, USA.
Email: monaghantf@gmail.com

Abstract

This study aims to determine whether dietary sodium restriction counseling decreases nocturnal voiding frequency in cardiology patients with concomitant nocturia. Patients who had established care at a cardiology clinic from 2015 to 2018 reporting ≥ 1 average nocturnal void(s) underwent a comprehensive sodium intake interview by their cardiologist, who provided them with individualized strategies for dietary sodium reduction and assessed adherence at follow-up. Average nocturnal voiding frequency and dietary adherence were documented in the medical record. A nocturia database was compiled for retrospective analysis. A total of 74 patients were included. Patients considered to be adherent with dietary sodium restriction at follow-up ($n = 56$) demonstrated a decrease in median nocturia frequency (2.5 [2.3-3.0] vs 1.0 [1.0-2.0] voids, $P < .001$). Among nonadherent patients ($n = 18$), median nocturia frequency did not significantly change from baseline to follow-up (2.0 [1.5-3.8] vs 2.0 [1.5-4.8] voids, $P = .423$). Median changes were significantly different between the adherent and nonadherent groups ($P < .001$). Examination of second follow-up available from 37 patients showed a continued effect. In conclusion, adherence with dietary sodium counseling appears to improve nocturia. Accordingly, dietary modification may represent an important adjunct therapy to lifestyle and pharmacologic interventions for decreasing nocturia frequency. Reduction in nocturnal voiding frequency may also reflect an additional benefit of dietary sodium restriction in accordance with best practice standards for cardiovascular disease.

1 | INTRODUCTION

Joint guidelines from the American Heart Association (AHA) and American College of Cardiology (ACC) endorse consideration of dietary sodium restriction in the management of hypertension and systolic heart failure, and recognize low sodium as an important non-pharmacologic lifestyle intervention for the primary prevention of

cardiovascular disease, with evidence suggesting that a diet low in sodium may decrease atherosclerotic cardiovascular disease (ASCVD) risk.¹⁻³ Among patients treated at a cardiology clinic, it has been our uncontrolled observation that sodium restriction also reduces nocturnal voiding frequency, even in those without hypertension or heart failure. However, to our knowledge, only one study has demonstrated an association between dietary sodium restriction and nocturia.⁴ Moreover,

patients enrolled in the aforementioned study were recruited from an inpatient setting, and it thus remains unclear whether dietary sodium restriction is an effective therapy for nocturnal voiding symptoms in cardiology practice. We hypothesized that sodium restriction would reduce nocturnal voiding frequency in patients who were adherent to dietary counseling recommendations. A secondary objective was to determine whether changes in nocturnal voiding frequency were associated with changes in blood pressure or weight.

2 | METHODS

Patients were evaluated at a university hospital-based outpatient cardiology clinic from July 2015 to September 2018. All patients were seen by a single cardiologist (JL) who performed a comprehensive sodium intake interview. A patient-centered approach to sodium restriction was employed. Upon dietary interview, specific sources of high dietary sodium were identified, and patients were provided individualized treatment plans for dietary sodium reduction.

At the time of follow-up, patients were considered to have been adherent or nonadherent (either did not significantly change or increase sodium intake). Both adherence and average self-reported nocturnal voiding frequency were recorded in the patient's medical record upon each visit. Nocturia was defined as waking to pass urine during the main sleep period, in accordance with current International Continence Society (ICS) terminology.⁵

A nocturnal voiding database was compiled for retrospective analysis upon approval from the SUNY Downstate Health Sciences University Institutional Review Board. All patient data were obtained from patient chart review. A waiver of informed consent was granted for retrospective analysis.

Change in voiding frequency was defined as the voiding frequency at follow-up minus the voiding frequency reported at the visit prior. The change in voiding frequency was compared in patients who had been adherent and nonadherent to dietary sodium advice at first and second follow-up encounters. Changes in weight and systolic and diastolic blood pressure from baseline to first follow-up were also compared for each patient.

Patients were included if they were ≥ 18 years of age and reported waking an average of 1 or more times per night in order to void at baseline. Patients were excluded if dietary sodium restriction was not warranted or contraindicated; if no follow-up data were available; or if baseline nocturia frequency, follow-up nocturia frequency, or follow-up dietary sodium adherence had not been clearly recorded in the patient's electronic medical record. Patients were also excluded if they had medication changes between visits. A total of 37 patients were followed for a second visit.

Several baseline variables, including basic clinical characteristics (age, sex, body mass index, ejection fraction, systolic blood pressure, diastolic blood pressure, nocturnal voiding frequency, and race), comorbid medical conditions (hypertension, hyperlipidemia, diabetes

mellitus, congestive heart failure, coronary artery disease, chronic obstructive pulmonary disease, chronic kidney disease, atrial fibrillation, aortic stenosis, mitral regurgitation, and aortic regurgitation), and diuretic utilization were also abstracted to further characterize the study population.

Categorical variables are reported as *frequency (%)*, and continuous variables are reported as *median (95% confidence intervals)* using Wilcoxon Confidence Interval estimates. Categorical variables were compared using Fisher's exact test. For continuous measures, the Wilcoxon signed-rank and Wilcoxon rank-sum tests were used to determine significance for paired and unpaired samples, respectively. A *P*-value of .05 was deemed statistically significant. Associations between the change in voiding frequency and changes in systolic blood pressure, diastolic blood pressure, and weight were assessed using Spearman's correlation coefficient.

3 | RESULTS

A total of 74 patients met the criteria for inclusion, of which 37 patients had two follow-up visits. Patient characteristics are provided in Table 1. By adherence status, diabetes mellitus was less prevalent at baseline among adherent as compared to nonadherent patients (34% vs 61%, $P = .041$).

The median interval was 50 (48-81) days from baseline to first follow-up and 88 (65-125) days from first follow-up to second follow-up. Study results for both first and second follow-up visits are summarized in Figure 1.

Among 74 patients, 56 were considered to be adherent with dietary sodium restriction, and these patients demonstrated a decrease in median nocturnal voiding frequency from 2.5 (2.3-3.0) to 1.0 (1.0-2.0) nocturnal voids ($P < .001$). The magnitude of this effect was similar in the subset of adherent patients with baseline diabetes mellitus ($n = 19$) (2.5 to 1.5 voids, $P < .001$). Among the 18 patients who were nonadherent, voiding frequency did not significantly change from baseline to follow-up (2.0 [1.5-3.8] to 2.0 [1.5-4.8] nocturnal voids [$P = .423$]). Median changes were significantly different between the adherent and nonadherent groups ($P < .001$).

Data for patients having two follow-up visits are portrayed in Figure 2. Patients adherent to salt restriction at either or both follow-ups ($n = 30$) were more likely to have a decreased number of voids when compared to patients who were nonadherent at both follow-ups ($n = 7$) ($P = .02$).

From baseline to first follow-up, there were no significant changes in median systolic blood pressure (137 [133-140] vs 136 [130-138] mm Hg, $P = .214$), diastolic blood pressure (76 [73-79] vs 74 [72-78] mm Hg, $P = .277$), or weight (85 [82-92] vs 85 [82-92] kg, $P = .712$). Change in nocturia frequency was not significantly correlated with either the change in systolic ($r = -.18$, $P = .139$) or diastolic ($r = -.19$, $P = .124$) blood pressure. Likewise, change in nocturia frequency was not significantly correlated with change in weight ($r = +.11$, $P = .386$).

TABLE 1 Baseline clinical characteristics

	All patients (n = 74)	Adherent (n = 56)	Nonadherent (n = 18)	P-value
Basic characteristics				
Age (y)	67 (63-69)	66 (62-70)	68 (63-73)	.687
Female//male	57 (77.0)//17 (23.0)	45 (80.4)//11 (19.6)	12 (66.7)//6 (33.3)	.230
Body mass index (kg/m ²)	31.7 (30.9-34.1)	31.6 (30.7-34.4)	31.8 (28.6-36.0)	.985
Ejection fraction (%)	62 (56-63)	62 (55-63)	64 (49-65)	.999
Systolic (mm Hg)	137 (133-140)	135 (130-139)	142 (135-150)	.075
Diastolic (mm Hg)	76 (74-79)	74 (72-78)	79 (73-86)	.165
Nocturia (average voids/night)	2.5 (2.3-3.0)	2.5 (2.3-3.0)	2.0 (1.5-3.8)	.320
Race				
Black/African American	69 (93.2)	52 (92.9)	17 (94.4)	.815
Hispanic	1 (1.4)	1 (1.8)	0 (0)	.568
Other/unspecified	4 (5.4)	3 (5.4)	1 (5.6)	.974
Comorbid conditions				
Hypertension	66 (89.2)	48 (85.7)	18 (100)	.090
Hyperlipidemia	34 (45.9)	24 (42.9)	10 (55.6)	.347
Diabetes mellitus	30 (40.5)	19 (33.9)	11 (61.1)	.041*
Congestive heart failure	30 (40.5)	21 (37.5)	9 (50.0)	.347
Coronary artery disease	17 (23.0)	11 (19.6)	6 (33.3)	.230
Chronic obstructive pulmonary disease	10 (13.5)	8 (14.3)	2 (11.1)	.732
Chronic kidney disease	7 (9.5)	4 (7.1)	1 (5.5)	.815
Atrial fibrillation	6 (8.1)	4 (7.1)	2 (11.1)	.592
Aortic stenosis	5 (5.8)	3 (5.4)	2 (11.1)	.398
Mitral regurgitation	3 (4.1)	3 (5.4)	0 (0)	.316
Aortic regurgitation	1 (1.4)	1 (1.8)	0 (0)	.568
Medications				
Loop diuretics	32 (43.2)	24 (42.9)	8 (44.4)	.906
Thiazide diuretics	15 (20.3)	12 (21.4)	3 (16.7)	.662

Note: Categorical variables reported as *frequency (%)*; continuous variables reported as *median (95% Confidence Intervals)*. (*) Denotes statistical significance.

4 | DISCUSSION

The major finding of the present study is that sodium restriction counseling in cardiovascular patients is accompanied by a significant reduction in nocturnal voiding frequency. A decreased number of voids was observed in patients who were considered to be adherent, but not in those who were not adherent. The difference in baseline

diabetes mellitus prevalence between adherent vs nonadherent patients did not appear to explain this effect. Changes in voiding frequency were not related to changes in blood pressure or to changes in weight. The present study results would suggest sodium intake to be an important determinant of nocturia in patients with various cardiovascular disease states, even in the absence of clinically evident volume overload. Accordingly, dietary modification would

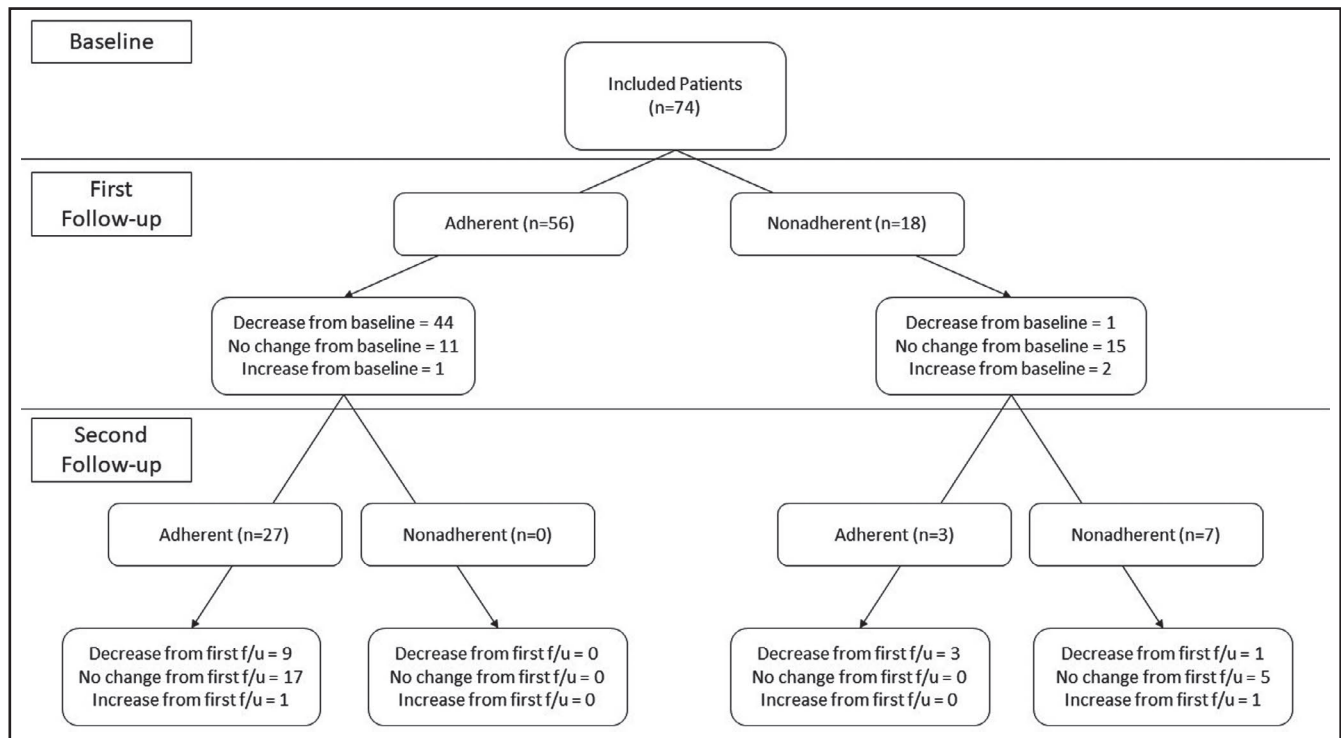


FIGURE 1 Self-reported patient adherence to individualized dietary sodium counseling provided at the baseline encounter. Second follow-up data were available for 37/74 (50%) patients included in the present analysis (29/56 [52%] of patients deemed adherent at first follow-up and 8/18 [44%] of patients deemed nonadherent at first follow-up were lost for second follow-up)

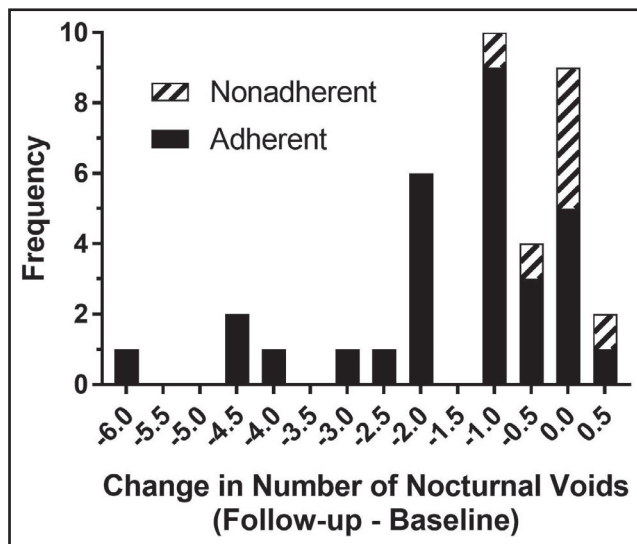


FIGURE 2 Frequency distribution of change in number of nocturnal voids (calculated as second follow-up minus baseline) for patients with two follow-ups. Patients adherent to sodium-restricted diet (at either first or second follow-up) ($n = 30$) shown in black; nonadherent patients at both follow-ups ($n = 7$) shown as cross-hatched. Difference significant at $P = .02$

appear to present an important adjunct therapy to reducing nocturia frequency in the setting of cardiovascular disease.

The present study results are consistent with and extend those reported by Matsuo and colleagues in inpatients.⁴ Among 321

inpatients recruited from a Japanese hospital, sodium restriction was associated with a decrease in nocturia in adherent patients, while patients who failed sodium intake restriction had no changes or worsening of nocturnal symptoms. Of note, the magnitude of change in the present study is similar to that found in the prior study and greater than for standard therapies for nocturia, including conservative lifestyle measures, behavioral therapy for chronic insomnia, appropriately timed diuretics, alpha blockers, and transurethral resection of prostate (TURP).⁶⁻⁹ Consistent with these findings are observational data that sodium restriction has been found to be an important determinant of nocturia. In a cross-sectional study of 728 patients with LUTS, excessive dietary sodium intake was identified as a risk factor for nocturia, which persisted after adjusting for age, sex, renal dysfunction, hypertension, diabetes mellitus, and hyperlipidemia.¹⁰

The cumulative results of these studies have important ramifications for cardiovascular disease patients. Nocturia has been found to be predictive of mortality, lower quality of life, and falls and hip fractures in the elderly, in whom cardiovascular risk factors/disease are prevalent.¹¹⁻¹⁵ In the setting of heart failure, Redeker and colleagues demonstrated that nocturia is independently associated with daytime fatigue, sleepiness, and impaired functional performance.¹⁶

While hypertension is the most common risk factor for congestive heart failure, it is notable that multiple studies have reported an association between nocturia and hypertension.^{17,18} Victor et al reported nocturia to be an important clinical clue of undiagnosed or uncontrolled hypertension in young black males.¹⁹ In the present

study, the lack of significant change in blood pressure with sodium restriction is consistent with the results of a prior meta-analysis.²⁰ It is possible that sodium restriction reduces nocturia frequency earlier than potential effects on blood pressure, which may lag. Accordingly, although nocturia is well known to be a symptom of congestive heart failure, its significance in this setting, and in other cardiac disease states, merits further study. It may be particularly illuminating to compare changes in nocturia severity with nocturnal blood pressure measurements through ambulatory blood pressure monitoring.

Several mechanisms may account for the findings of this study, including subclinical volume overload, more favorable sodium balance with sodium intake not exceeding sodium excretion, and lower levels of plasma brain natriuretic peptide, which has been implicated in the pathogenesis of nocturia.^{21,22} The precise mechanisms by which cardiovascular patients with concomitant nocturia respond to dietary sodium restriction warrant further investigation. Namely, objective indices of renal function, electrolyte balance, and fluid status, including blood and urinary sodium concentrations, plasma natriuretic peptide levels, and peripheral edema (eg, impedance measurement), are needed to explore potential mechanisms underlying the results described in the present study.

The present investigation is subject to the inherent limitations of a retrospective study. Notably, subjective assessment of dietary sodium intake is less reliable and more susceptible to observer bias and under-reporting than 24-hour urine collection.^{23,24} In view of inherent differences in provider knowledge and attitudes surrounding dietary sodium and nutrition, other methods of assessing dietary sodium intake, such as dietary questionnaires, may be more feasibly standardized and generalized for use in routine clinical practice. Data pertaining to sleep duration and timing of nocturia were not collected. We evaluated weight, but not peripheral edema, which is difficult to assess with regard to severity and extent. We used voiding frequency as a measure of the severity of nocturia rather than voiding diaries, which are considered the gold standard for the evaluation and management of LUTS.²⁵ Further limitations include single institution and provider participation, small sample size, and a relatively high proportion of African Americans, who have been previously reported to be relatively more salt-sensitive with regard to blood pressure elevation.²⁶

Adherence with dietary counseling aimed at sodium restriction significantly reduces nocturnal voiding frequency in a manner that is unrelated to changes in blood pressure and weight. However, nocturia is a complex and oft-multifactorial condition that has been associated with a number of cardiovascular disease states, but may also be a harbinger of a wide range of serious endocrine, metabolic, hepatic, renal, immune, and neurologic disorders, or rather be the direct result of an abnormality in the genitourinary tract.²⁷ Furthermore, the pathogenesis of nocturia may be confounded by sleep disorders, concurrent medications, and various modifiable lifestyle factors in addition to dietary sodium (eg, water, caffeine, and alcohol intake).²⁸ Accordingly, future prospective trials with well-defined nocturia patient subgroups and objective outcome indices will be needed to further characterize the relationship between nocturia and dietary

sodium intake, elucidate potential mechanisms, and establish the direct clinical implications of dietary sodium restriction in relation to nocturnal voiding.

5 | CONCLUSION

In the present study of patients seen at a cardiology clinic, a significant decrease in nocturnal voiding frequency was observed in those deemed to be adherent to individualized sodium dietary counseling from their cardiologist. Our results suggest that reduction in nocturnal voiding frequency may represent an additional benefit of maintaining dietary sodium in accordance with best practice standards in the evaluation and management of cardiovascular disease. Notably, change in voiding frequency was not significantly correlated with change in blood pressure. Further research is needed to establish the etiology of nocturia response and quantify the relationship between sodium intake and nocturia in this patient population.

CONFLICT OF INTEREST

Thomas F. Monaghan has no direct or indirect commercial incentive associated with publishing this article and certifies that all conflicts of interest relevant to the subject matter discussed in the manuscript are the following: Dr Bliwise has served as a consultant for Merck, Jazz, Ferring, Eisai, and Respicardia and speaker for Merck within the last 3 years, outside the submitted work. Dr Everaert is a consultant and lecturer for Medtronic and Ferring and reports institutional grants from Allergan, Ferring, Astellas, and Medtronic, outside the submitted work. Dr Vande Walle reports institutional grants from Allergan, Astellas, and Ferring, and is a consultant and lecturer for Ferring and Astellas, outside the submitted work. Dr Weiss is a consultant for Ferring and the Institute for Bladder and Prostate Research, outside the submitted work. The other authors have nothing to disclose.

AUTHOR CONTRIBUTIONS

Monaghan, Weiss, and Lazar contributed to study conception and design. Monaghan, Michelson, Wu, Gong, Agudelo, George, Mekki, and Lazar contributed to acquisition of data. Monaghan, Alwis, Bliwise, Weiss, and Lazar contributed to analysis and interpretation of data. Monaghan and Lazar contributed to drafting of manuscript. Monaghan, Michelson, Wu, Gong, Agudelo, George, Alwis, Epstein, Flores, Bliwise, Everaert, Vande Walle, Weiss, and Lazar contributed to critical revision.

ETHICAL APPROVAL

A nocturnal voiding database was compiled for retrospective analysis upon approval from the SUNY Downstate Health Sciences University Institutional Review Board. A waiver of informed consent was granted for retrospective analysis.

ORCID

Thomas F. Monaghan  <https://orcid.org/0000-0002-5236-0783>

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